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NR3C1 Blocking Peptide (C-term)

Synthetic peptide
Catalog # BP19840b

Specification

NR3C1 Blocking Peptide (C-term) - Product Information

Primary Accession	P04150
Other Accession	P59667, Q9N1U3, P06537, NP_000167.1

NR3C1 Blocking Peptide (C-term) - Additional Information

Gene ID 2908

Other Names
Glucocorticoid receptor, GR, Nuclear receptor subfamily 3 group C member 1, NR3C1, GRL

Target/Specificity
The synthetic peptide sequence is selected from aa 575-588 of HUMAN NR3C1

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

NR3C1 Blocking Peptide (C-term) - Protein Information

Name NR3C1 (HGNC:7978)

Synonyms GRL

Function
Receptor for glucocorticoids (GC) (PubMed:27120390). Has a dual mode of action: as a transcription factor that binds to glucocorticoid response elements (GRE), both for nuclear and mitochondrial DNA, and as a modulator of other transcription factors (PubMed:28139699). Affects inflammatory responses, cellular proliferation and differentiation in target tissues. Involved in chromatin remodeling (PubMed:9590696). Plays a role in rapid mRNA degradation by binding to the 5' UTR of target mRNAs and interacting with PNRC2 in a ligand-dependent manner which recruits the RNA helicase UPF1 and the mRNA-decapping enzyme DCP1A, leading to RNA decay (PubMed:25775514). Could act as a coactivator for STAT5-dependent transcription upon growth hormone (GH) stimulation and could

reveal an essential role of hepatic GR in the control of body growth (By similarity).

Cellular Location
[Isoform Alpha]: Cytoplasm. Nucleus. Mitochondrion. Cytoplasm, cytoskeleton, spindle. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Note=After ligand activation, translocates from the cytoplasm to the nucleus. In the presence of NR1D1 shows a time-dependent subcellular localization, localizing to the cytoplasm at ZT8 and to the nucleus at ZT20 (By similarity). Lacks this diurnal pattern of localization in the absence of NR1D1, localizing to both nucleus and the cytoplasm at ZT8 and ZT20 (By similarity). {ECO:0000250|UniProtKB:P06537, ECO:0000269|PubMed:18838540, ECO:0000269|PubMed:27120390, ECO:0000269|PubMed:8621628} [Isoform Alpha-B]: Nucleus. Cytoplasm Note=After ligand activation, translocates from the cytoplasm to the nucleus.

Tissue Location
Widely expressed including bone, stomach, lung, liver, colon, breast, ovary, pancreas and kidney (PubMed:25847991). In the heart, detected in left and right atria, left and right ventricles, aorta, apex, intraventricular septum, and atrioventricular node as well as whole adult and fetal heart (PubMed:10902803) [Isoform Alpha-2]: Widely expressed.

NR3C1 Blocking Peptide (C-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

NR3C1 Blocking Peptide (C-term) - Images

NR3C1 Blocking Peptide (C-term) - Background

The protein encoded by this gene is a receptor for glucocorticoids that can act as both a transcription factor and as a regulator of other transcription factors. This protein can also be found in heteromeric cytoplasmic complexes along with heat shock factors and immunophilins. The protein is typically found in the cytoplasm until it binds a ligand, which induces transport into the nucleus. Mutations in this gene are a cause of glucocorticoid resistance, or cortisol, resistance. Alternate splicing, the use of at least three different promoters, and alternate translation initiation sites result in several transcript variants encoding the same protein or different isoforms, but the full-length nature of some variants has not been determined.

NR3C1 Blocking Peptide (C-term) - References

Seitz, T., et al. J. Mol. Biol. 403(4):562-577(2010)
Wang, W., et al. Nucleic Acids Res. (2010) In press :
Sarzynski, M.A., et al. Int J Obes (Lond) (2010) In press :
Desrivieres, S., et al. Addict Biol (2010) In press :
van Oosten, M.J., et al. Arthritis Res. Ther. 12 (4), R159 (2010) :